FLUID-STRUCTURE INTERACTION ANALYSIS OF HUMAN HEART BY ALE FINITE ELEMENT METHOD

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The heart, especially the left ventricle (LV), plays a central role in the circulation of blood; thus, it is one of the essential organs for supporting human life. The motion of LV consists of the following four phases: Phase 1 – isovolumic contraction period, phase 2 – ejection period, phase 3 – isovolumic relaxation period, and phase 4 – filling period. At the end of ejection, the ventricular volume is about 40 to 45 percent of the volume at the beginning of ejection.

To construct a comprehensive model of heart which adequately simulates the events described above, microscopic as well as macroscopic mechanism should be incorporated. Several models of electrical activity of cardiac cells were proposed, and large-scale electrophysiological computer simulations of heart have been performed recently. The research field of myocardial excitation-contraction coupling has been explored by physiologists. At the macroscopic level, the motions of LV were simulated by solid finite elements. The intra-ventricular blood flow has been simulated by many researchers, and recently the fluid-structure interaction analyses of heart were done with immersed boundary methods by Lemmon and Yoganathan[1], and McQueen and Peskin[2]. However, none of them simulated the blood flow and wall motion based on the microscopic physiological models.

The objective of this study is to develop a fluid-structure interaction (FSI) finite element based simulation program incorporating the propagation of excitation and contraction mechanisms of cardiac cells to simulate the pumping action of human LV. An arbitrary Lagragian-Eulerian (ALE) finite element method with automatic mesh updating is formulated for large domain changes, and a strong coupling strategy[3] is taken. The Luo-Rudy model is used for the excitation and propagation models, and the four-state model proposed by Negroni et al. is employed for excitation–contraction coupling. A constitutive equation for cardiac muscles proposed by Lin and Yin is employed. In the simulation of normal excitation, the propagation of excitation and the intra-ventricular flow pattern are in good agreement with the clinical observation. The wall deformation, stress and strain are also investigated. As an example of arrhythmia, extrasystole is simulated. The propagation of excitation and the intra-ventricular flow pattern and the P-V loops well reproduced the clinical observation that reflects the insufficient filling and asynchronous excitation.

References

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